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Case Report

Post-operative hyperkalaemic paralysis

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Tetraparesis is more often a clinical feature of profound hypokalaemia [1, 2] than hyperkalaemia. Neurological features of hyperkalaemia are rarely seen in clinical practice, as they are precluded by cardiotoxic complications such as arrhythmias. However, patients may develop flaccid paralysis of skeletal muscle with areflexia mimicking symptoms of acute inflammatory demyelinating polyneuropathy.[3] Severe hyperkalaemic paralysis has previously been reported secondary to nonsteroidal anti-inflammatory drugs [4] spironolactone,[5] and a combination of chronic renal failure and ACE inhibitors.[6] We report a case of post-operative secondary hyperkalaemic paralysis presenting with neurological symptoms 18 days after an anterior resection (with diverting loop ileostomy) for a rectosigmoid colonic adenocarcinoma.

CASE REPORT A 57-year-old man was referred to the colorectal clinic with symptoms of change in bowel habit, colicky central abdominal pain and weight loss. He had a history of maturity onset diabetes mellitus, hypertension and peripheral vascular disease having previously had a right femoral angioplasty. Medication consisted of metformin, gliclazide, amlodipine, atorvastatin, aspirin and lisinopril combined with a thiazide diuretic. A double contrast barium enema and flexible sigmoidoscopy revealed a polypoid lesion at the rectosigmoid junction. Histology of representative biopsies confirmed endoscopic suspicion of an adenocarcinoma. He underwent a technically difficult anterior resection with formation of a defunctioning loop ileostomy. Histopathology reported a Dukes' Cl (PT3 N1 Mx) rectal adenocarcinoma, which extended to within 1mm of the circumferential margin. Apart from poor post-operative glycaemic control necessitating an increased dose of metformin, he made a relatively uneventful recovery and was discharged home on the twelfth post-operative day, confident with ileostomy management. The electrolytes were normal at the time of discharge.

After 48 hours he developed increased ileostomy effluent output followed by symptoms of anorexia, nausea then vomiting. By the following day he was feeling generally tired and weak. He continued to deteriorate over the next 48 hours, and became bedbound. There were however no bulbar or respiratory symptoms.

On readmission clinical examination revealed that he was afebrile but markedly dehydrated and weak, hypotensive (75/50 mmHg) with a heart rate of 60 beats/min and respiratory rate of 14 breaths/min. Capillary blood glucose was 18 mmol/L. Abdominal and respiratory examination were unremarkable. Glasgow coma scale was 15/15, there was no facial asymmetry and other cranial nerves were unaffected. Neurological examination of the limbs revealed profound weakness of all four limbs, particularly distally (MRC grade 1-2/5). He was areflexic and generally flaccid. There was no sensory deficit and plantar responses were bilaterally flexor. The differential diagnosis was wide (Table).

Blood results at the time of admission revealed a serum potassium of 8.8mmol/L (confirmed on repeat sample), urea 45.8mmol/L, creatinine

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TABLE
Differential diagnosis

Acute inflammatory demyelinating polyneuropathy (AIDP)
Metabolic neuropathy/myopathy
Carcinomatous neuropathy/myopathy
Lambert-Eaton myasthenic asyndrome
Critical illness neuropathy/myopathy

644umol/L and a compensated metabolic acidosis (base excess -9.5). An electrocardiogram showed T wave 'tenting' consistent with hyperkalaemia. Calcium gluconate was administered intravenously with oral calcium resonium subsequently. Two consecutive infusions of 50% dextrose and insulin were administered with normal saline rehydration resulting in resolution of the acidosis and improvement in the electrolytes. Within hours of treatment neurological symptoms resolved and muscle power returned. Urea and creatinine fell to normal levels over course of one week. He was discharged 9 days after readmission and has remained well.

DISCUSSION

Hyperkalaemic paralysis can be primary or secondary. Patients with primary episodic or hereditary hyperkalaemic periodic paralysis have a genetically determined defect in the sodium channels of muscle fibre membranes (channelopathy).[7] Typically the clinical onset is in the first decade of life with attacks of flaccid paralysis lasting from a few minutes to several hours, at intervals of hours to days. Attacks are usually induced by the ingestion of potassium and by rest after exercise. Affected individuals may exhibit the ability to "walk off' an attack and can be successfully treated by thiazide diuretics, acetazolamide and salbutamol.

Secondary hyperkalaemic paralysis occurs later in life and is caused by a partial defect in the sodium channels of muscle fibre membranes, however the precise mechanism is unknown. The potassium may have either a direct effect on the muscle cell membrane or possibly disturb the peripheral nerves supplying the muscle.[7] It usually occurs in elderly patients with underlying chronic renal impairment, but can be precipitated by drugs[4,5,6] or trauma.[8] Flaccid paralysis with areflexia (initially affecting the legs and progressing to the arms) with preservation of sensation and cranial nerve function are signs common to both hyperkalaemic paralysis and acute inflammatory demyelinating polyneuropathy (Guillain-Barré syndrome). Paralysis can be so severe and progressive as to involve respiratory muscles necessitating artificial ventilation.[9] This also has implications for the anaesthetist's choice of muscle relaxant prior to intubation. A depolarising agent such as suxamethonium may further increase serum potassium precipitating a fatal arrhythmia.

The largest cohort of cases of secondary hyperkalaemic paralysis in the literature suggests that the clinical presentation and subsequent clinical course in our patient is typical of this condition. Of the 18 cases described by Evers *et al*,[15] presented with tetraparesis/tetraplegia and three with paraparesis. Paralysis typically began distally with an ascending course and although significant sensory signs were not found, sensory symptoms were reported in five cases. The mean serum potassium concentration at presentation was 9.0 mmol/L, two-thirds of cases had chronic renal impairment and 10/18 cases were precipitated by potassium-sparing diuretics. The generally favourable prognosis of the condition is emphasised by 15/18 patients making a good recovery within hours to days, although the potentially serious consequences are also highlighted by the two deaths due to cardiac arrest.[10] Our patient had a major colorectal operation 18 days before becoming paralysed. An ACE inhibitor in combination with increased ileostomy output leading to dehydration caused progressive renal impairment, leading to hyperkalaemia and secondary paralysis. Progressive weakness and physical inability to drink exacerbated the dehydration.

The prognosis for secondary hyperkalaemic paralysis is good if recognised and treated. Clinicians should be aware of this possible complication in post-operative patients with electrolyte disturbance secondary to increased stoma effluent.

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